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MASS SELECTION FOR BLISTER RUST RESISTANCE: A METHOD FOR NATURAL REGENERATION OF WESTERN WHITE PINE

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ABSTRACT

Seeds from surviving western white pine in stands that have sustained high (80 to 90 percent) mortality from blister rust produce 19.8 percent healthy seedlings--an 18 percent increase in resistance over that of the native population prior to introduction of blister rust. A breeding approach that would use this natural increase of resistance is proposed.

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White pine blister rust, caused by *Cronartium ribicola* J.C. Fisch. ex Rabenh., was first observed in the inland population of western white pine (*Pinus monticola* Dougl.) in 1927. Infection, which dated back to 1923 (Mielke 1943), spread slowly but steadily until 1940 when incidence increased rapidly. At present, rust occurs throughout the range of western white pine. Tree size and the amount of infection determine the swiftness and extent of mortality in the stand (Mielke 1943). Stands exhibiting low mortality are characteristically composed of overmature (150- to 200-year-old) white pine or are located where conditions are less favorable for blister rust infection. In the majority of the inland white pine populational zones, temperature and moisture conditions are near optimal for infection, and tree losses have been extensive.

As the infection level increases, a few trees remain free of disease or relatively so compared to adjacent trees. In 1950, the USDA Forest Service started a

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program to determine whether this phenotypic resistance is heritable. High amounts of resistance and a large number of resistance mechanisms have been found (Bingham and others 1971). Although, to date, we probably have detected only a few of the possible resistance mechanisms and underestimated the number of genes present (table 1) (there must be many, even 20-30 or more), we are rapidly incorporating this resistance into planting stock. Five seed orchards have been established and others are planned. The aim is to maximize the level of resistance and the number of resistance mechanisms as well as to maintain adaptability. Data pertinent to this program are presented in Steinhoff (1971), Bingham and others (1971; 1973), and Hoff and others (1973).

Since most man-guided breeding programs seem to yield populations that eventually lose much of their adaptability (National Research Council 1972), we can expect that the seed orchard approach could lead to populations deficient in adaptability. Loss of adaptability would be intolerable in the case of white pine, a long-rotation (80-year) crop, since the new population will be growing in a natural or seminatural situation under continual environmental stress from weather, insects, other diseases, and heavy competition from associated species.

Table 1.--*Observed resistance mechanisms in* Pinus monticola:Cronartium ribicola *system*

Mechanism of resistance	Hypothesized inheritance
Resistance in secondary needles to a yellow spot-forming race	Recessive gene
Resistance in secondary needles to a red spot-forming race	Dominant gene
Resistance in secondary needles to a yellow-green-island spot-forming race	Dominant gene ?
Resistance in secondary needles to a red-green-island spot-forming race	Dominant gene ?
Reduced frequency of secondary needle infections	Nondominant gene ?
Slow fungus growth in secondary needles	Polygene ?
Premature shedding infected secondary needles	Recessive gene
Fungicidal reaction in short shoot	Recessive gene
Fungicidal reaction in stem	Oligogene ?
Slow fungus growth in stem	Polygene ?
Tolerance to infection	?

The purpose of this paper is to discuss recurrent mass selection in natural stands, a breeding approach that could yield genetically diverse seed crops characterized by stable resistance and broad adaptability. The selection force would be the natural levels of blister rust inoculum. We think mass selection would yield the desired breadth of resistance and adaptability; infected white pines that remain in stands exhibiting high mortality appear to contain relatively high levels of resistance, and large numbers of parent trees are available to assure broad adaptability. Although natural processes would eventually solve the problem, they are often unsuitable and lengthy. We hope that judicious guidance of natural processes will accelerate development of a new population of resistant, broadly adapted white pine.

STABILITY OF RESISTANCE

The most frequent question asked about upcoming populations of rust-resistant western white pine produced naturally or by artificial breeding methods, is, "Will new races capable of neutralizing the resistance genes arise?"

Certainly races will arise; we have already seen at least two (McDonald and Hoff in press). It is necessary to assume that the rust is highly adaptable and will continue to evolve races for the breeder to contend with. Recombinations occur every year and can be disseminated by fruiting cankers, most of which are long lived. Then, too, pine infections are haploid; so each new recombinant, mutation, or both, can be immediately expressed.

So how can we stabilize the resistance in a new population? New races commonly arise soon after release of a new variety (van der Plank 1968; National Research Council 1972). The problems caused by breeding disease-resistant wheat, corn, potatoes, and other crops are well known. Tired of having their efforts neutralized time after time, the breeders developed a new breeding philosophy based on the coexistence of host and parasite. Diversity is the objective of most recent breeding schemes that should stabilize resistance in crop plants (van der Plank 1968; Watson 1970; Leppik 1970; National Research Council 1972; and others). This concept agrees with nature's method--diversity is the rule in natural ecosystems (Browning 1975). Accordingly, we believe that diversity, developed and maintained by a more natural breeding method, can best stabilize resistance to blister rust in western white pine.

Mass selection will provide a high degree of genetic diversity and resistance stability because the total array of resistance will be selected--not just those traits man can see; the total genetic diversity of the rust will be used as inoculum--not just the sample man picks; and all environments can be included in the selection--not just the few to be used in expensive artificial breeding programs.

Mass selection for blister rust resistance has been going on from 30 to 40 years--ever since blister rust killed the first white pine. Natural mortality in some white pine stands has reached 80 to 90 percent. Trees remaining in these stands have been mass selected for resistance to blister rust.

Resistance in White Pine Prior to Introduction of Blister Rust

Early workers recognized that western white pine was susceptible to blister rust (Spaulding 1929). In British Columbia, all but 9 of 500 trees planted in 1923 had been killed by rust by 1929 (Mielke 1943). A systematic sample of a square mile of heavily infected 40- to 50-year-old white pine disclosed that only 1 of 384 trees was free of living blister rust cankers (Bingham 1947). Bingham and others (1972) described a younger stand where only 1 of about 10,000 trees was completely free of living or dead cankers. In 1952, five seed lots were sown as controls in a test of

blister rust resistance in progeny. The seed lots were collected from squirrel caches in five northern Idaho-northwestern Montana stands where rust mortality then ranged from almost none to about 15 percent. Control seedlings were artificially inoculated the fall of their second growing season and outplanted on three field plots where they have since received heavy natural inoculation. Annual examinations of individual trees were made from 1954 to 1958, and periodic examinations were conducted in 1966 and 1973. By 1973, 98 percent of the 371 control trees had been killed by the rust; 8 trees (2 percent) remained alive, but only 3 of these (0.8 percent) were free of living blister rust cankers. Hence, the original population of inland western white pine was highly susceptible to blister rust; only about 1 or 2 percent of the individuals in it were resistant under heavy rust exposure.

Resistance of Surviving White Pine

The pertinent question is, how much and what kind of resistance will the survivors in stands that have had high (80 to 90 percent) mortality transmit to their progeny? One answer is available in the control lots used in later (1964-66) tests of progeny chosen for resistance to blister rust. In these tests, control lots were from areas where mortality was high. These lots came from squirrel caches or from collections of wind-pollinated seed produced by infected trees.

Nearly 20 percent of the progeny from these control lots were healthy 2.5 to 4 years after inoculation (table 2). Compared to the recorded resistance level prior to severe selection by blister rust, this is an 18 percent increase. Seed lots from stands with little or no mortality were not included; so a good comparison was not possible. On the other hand, all data were derived from seedlings that had needle spots 9 months after inoculation, and we feel that the resistance indicated is conservative. Seedlings without needle spots were eliminated; this usually amounted to less than 2 percent.

Causes of the considerable year-to-year variation within seed collections are unknown. Possible explanations are variation in inoculation conditions, in racial composition of the inoculum, or in environmental influence on the expression of resistance. Nevertheless, the general level of resistance of all control collections is relatively high over the 3 test years.

The resistance shown in table 2 is made up of three resistance mechanisms:

1. *Premature shedding of infected needles.* Infected needles drop off before the fungus can enter the stem; therefore, seedlings with this trait remain healthy. This mechanism apparently is inherited as a single recessive gene (McDonald and Hoff 1970).

2. *Fungicidal reaction in the short shoot.* This reaction is initiated by the interaction of the fungus and tissues around the short shoot that results in the death of the fungus. This reaction is also thought to be inherited as a single recessive gene (Hoff and McDonald 1971; McDonald and Hoff 1971).

3. *Fungicidal reaction in the stem.* This reaction is the result of an interaction of the fungus and stem tissue that produces large patches of dead host tissue. Although the fungus is not killed, it is slowed or starved by the necrosis. This reaction is followed shortly by the formation of wound periderm, which can act as a barrier between healthy and infected host tissues. Inheritance of this reaction seems to be controlled by several genes (Struckmeyer and Riker 1951; Bingham and others 1960; Hoff and McDonald 1972).

Table 2.--*The level of resistance of seed lots collected from rust-infected western white pine stands with high mortality*

Control lot	Progeny test: 1964 (4 yr after inoc.) Uninfected plants	Total plants	Percent uninfected plants	1965 (3 yr after inoc.) Uninfected plants	Total plants	Percent uninfected plants	1966 (2.5 yr after inoc.) Uninfected plants	Total plants	Percent uninfected plants
DD[a]	12	85	14.1						
NN[a]	22	78	28.2						
OO[a]	10	88	11.4				4	40	10.0
SS[b]	17	69	24.6						
WW[b]	34	81	42.0	38	204	18.6	29	138	21.0
QQ[b]	11	37	29.7						
RR[a]	11	60	18.3	43	195	22.1	0	41	0
VV[b]	27	82	32.9				5	49	10.2
YY[b]	6	78	7.7						
XX[b]	14	61	23.0	13	121	10.7	36	145	24.8
TT[a]				23	150	15.3	29	133	21.8
ZZ[b]				28	65	43.1			
NR11 x Mix[c]				9	64	14.1			
NR12 x Mix[c]				1	10	10.0			
NR13 x Mix[c]				2	64	3.1			
NR14 x Mix[c]				3	17	17.6			
Total	164	719	22.8	160	890	18.0	103	546	19.0
Grand total	427	2,155	19.8						

[a]Collections from five wind-pollinated, rust-infected trees.
[b]Collections from squirrel caches.
[c]Crosses made on specific trees with a mix of pollen from 10 rust-infected trees.

Table 3 shows the frequencies of these three mechanisms of resistance. For comparative purposes, we have included data from the F_1 and F_2 crosses of resistant candidates and their progeny. Compared to the level of resistance in the original populations, considerable selection for these resistance mechanisms has taken place. Although we have partitioned resistance into three categories, two of which are presumably controlled by single genes, we have no data concerning the total number of resistance mechanisms nor of genes present in these surviving trees. An extensive study is underway that is expected to provide answers to these questions.

Table 3.--*Percentage and gene frequencies of various mechanisms of resistance in western white pine to infection by* C. ribicola

Seed lots	Recessive gene 1, premature needle shedding	g.f.	Recessive gene 2, fungicidal short shoot	g.f.	Bark reactions	Rust-free plants	Total seedlings
	Percent						
1st Progeny Test							
Susceptible[a]	7.6	0.24	[b]5.0	0.12	[b]14.6	22.8	719
F1[c] (52 candidates X 4 testers)	7.0	.28	[b]20.4	.45	[b]24.1	39.5	16,046
2nd Progeny Test							
Susceptible	14.3	0.38	[b]2.4	0.15	[b]2.9	18.9	546
F1[d] (10 families)	19.9	.45	[b]6.5	.25	[b]10.2	32.7	2,876
F1[e] (52 families)	47.4	.69	[b]21.9	.47	[b]16.8	65.8	3,061

[a]Wind-pollinated seed from infected parents in stands with high mortality.
[b]Values adjusted for previous mechanisms of resistance.
[c]Two parent crosses of individuals without cankers selected as phenotypically resistant candidates.
[d]Families from previous progeny tests that have shown uniformly high general resistance (Bingham and others 1960).
[e]Two parent crosses of resistant seedlings, derived from families in previous progeny tests, that have shown uniformly high general resistance.

THE NUMBERS GAME

Stands exhibiting high mortality still contain many living trees. Four stands that we have been investigating average 46 living trees per acre. According to Bingham and Rehfeldt (1970), trees 45 to 60 years of age will average 28 cones per year and 120 seeds per cone--3,360 seeds per year per individual tree. Haig and others (1941) reported that about 2 percent of the annual seed production will result in established seedlings (a living seedling 6 years after germination); therefore, each individual tree is expected to produce 67 seedlings per year. After disturbance due to fire or clearcutting, natural regeneration can occur successfully for 5 to 10 years (Haig and others 1941). Over a 5-year period one tree should produce 335 seedlings. If a stand that has shown high mortality averages 46 living trees per acre (table 4), 15,410 established seedlings can logically be expected. If 19.8 percent of these seedlings were resistant (table 2), each acre could end up with 3,051 healthy trees (table 5).

Table 4.--*Total living western white pines per acre in four high-mortality stands*

Stand	Tree total	Total living	Mortality
			Percent
Bertha Hill	390	70	82
White Rock Springs	190	48	79
Elk Creek	182	20	89
Pete's Creek	434	46	89
Average	299	46	88

Table 5.--*Potential numbers of resistant white pine seedlings that could be produced per acre through mass selection in stands with high mortality*

Cones per tree	28
Seeds per cone	120
Seeds per tree	3,360
Established seedlings (2 percent of seed in 6 years)	67
Years for natural regeneration, 5 to 10; choose 5 years	5
Established seedlings per tree	335
Average living trees per acre	46
Total established seedlings per acre	15,410
Percent resistance	19.8
Established resistant seedlings per acre	3,051

PRACTICAL USE OF MASS SELECTION FOR RESISTANCE TO BLISTER RUST

The beauty of using mass selection of naturally selected resistant western white pine is its simplicity. It can easily be incorporated into existing practices. Some foresters, exercising their biological intuition, have already used this approach. They believe that healthy, living white pines remaining after the rust has killed most of the trees in a stand must contain some resistance. We agree.

Our general recommendations for using mass selection are as follows:

1. Select contrasting sites that once supported good white pine stocking that has been reduced 80 to 90 percent by blister rust;

2. Remove dead or high-risk white pine (table 6) and other competing trees or shrubs;

3. Prepare a seedbed. The most economical practice would be to prepare the selected site during logging;

4. Remove seed trees from each site after adequate regeneration--at least 2,000 seedlings per acre.

The new population can be managed as a timber stand to produce fiber, as a seed production area to yield resistant white pine seed, or as both. After infection and mortality of the most susceptible seedlings, the level of resistance is expected to be 50 to 60 percent. This estimate is based upon data produced by our artificial breeding program (Bingham and others 1973; Hoff and others 1973).

If several seed production areas are established in the many contrasting environments throughout the inland range of western white pine, we would expect adaptability to be optimum because seedling selection will have taken place on natural sites by natural processes.

Table 6.--*Condition and rate of mortality of surviving western white pine trees in stands that have sustained various levels of blister rust mortality*

Condition	<10 percent			50 percent			>38 percent		
	Condition[b] of individuals in 1970	Trees dead by 1974	Condition of individuals in 1974	Condition of individuals in 1970	Trees dead by 1974	Condition of individuals in 1974	Condition of individuals in 1970	Trees dead by 1974	Condition of individuals in 1974
Excellent[a]	49	0	44	69	0	69	77	3	56
Good	1	0	4	17	1	3	30	4	19
Fair	0	0	2	10	2	9	16	5	18
Poor	0	0	0	3	2	6	8	3	12
Very poor	0	0	0	1	1	7	19	13	17
Dead (total)	0	0	0	0	6	6	0	28	28

[a]This is a visual rating class of the general appearance of the parent trees; blister rust cankers are ignored.

[b]First rating was completed in 1971, but, since cones were collected from all trees in 1970 and because changes are slight between years, we assumed the 1971 ratings would closely reflect conditions in 1970.

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